Available online at www.sciencedirect.com

SciVerse ScienceDirect

Procedia - Social and Behavioral Sciences 32 (2012) 425 – 430

Procedia
Social and Behavioral Sciences

4th International Conference of Cognitive Science (ICCS 2011)

Comparison of amplitude and latency of cognitive potential (P3) with high and low frequency stimuli in early and late onset blind subjects

Nasim Manouchehri[a,*], Mansoureh Adel Ghahraman[a], Saied Farahani[a]

[a]*Faculty of Rehabilitation, Tehran University of Medical Sciences, Tehran, Iran*

Abstract

The aim of this study was to study P3 with high and low frequency stimulus in blind subjects to study neuroplasticity. The P3 was recorded in 70 dBnHL. Comparison of results between two groups showed that there were no statistically significant differences between amplitudes and latencies of P3 with high frequency stimuli. However, in low frequency the difference between amplitudes was significant. Neuroplasticity seemed to occur in visual cortex in both groups. The activation patterns of the occipital cortex varied between these two groups and this could be demonstrated with low frequency stimuli better than with high frequency stimuli.

Keywords: Cognitive potential; blindness; neuropalsticity; P3, reorganization

1. Introduction

The P3 potential was first reported by Davis (1965) and Sutton and colleagues (1965). This response is elicited in normal subjects in 250-400 ms after stimulus onset and is conventionally recorded with oddball paradigm. Since the presence of the P3 response depends on subject's attention to rare signal, it is often described as a cognitive potential (Hall, 2007).

During childhood development, neural connections are formed in the brain area that is dependent on afferent input. Animal studies have shown cortical area responses to auditory and somatosensory stimuli after visual deprivation that called Cross-modal plasticity (Theoret, Merabet, & Pascual-Leone, 2004; Maurer, Lewis, & Mondloch, 2005; Stevens & Weaver, 2009). Otherwise, human studies with ALR have shown that congenital blind subjects have more sensitive auditory perception (Naveen, Srinivas, Nirmala, Nagendra, & Telles, 1997). Auditory compensation in blind subjects is not clearly known. Blind subjects have better function in some sophisticated auditory function than sighted subjects that demonstrate that compensation occurs at higher and more central level of data processing in blindness (Alho, Kujala, Paavilainen, Summala, & Naatanen, 1993; Roder, Rosler, & Neville, 2000; Burton, Diamond, & McDermott, 2003a). Electrophysiological studies in event-related potentials have

* Corresponding author. Tel.: +98-(261)2754361; fax: +98-(261)2754361
E-mail address: nmanoochehri@razi.tums.ac.ir, nasim_manoochehri@yahoo.com

1877-0428 doi:10.1016/j.sbspro.2012.01.064

indicated that the blind subjects produced larger P3 amplitudes and shorter latencies than sighted subjects (Roder, Rosler, Hennighausen, & Nacker, 1996; Kujala, Alho, & Naatanen, 2000). In addition, Neuroimaging studies have also indicated more activity of posterior areas primary visual cortex along non-visual tasks such as auditory or somatosensory in blind subjects compare with sighted subjects that probably related to cross-modal plasticity in blind subjects (Alho et al., 1993; Leclerc, Staint-Amounr, Lavoie, Lassonde, & Lepore, 2000).

Niemeyer and Starlinger (1981) and Woods, Clayworth, and Bach-Y-Rita, (1985) reported that congenital blind subjects have significantly shorter N1, P2 (ALR) latencies and larger amplitude than sighted subjects. This study also represented that visual cortex in congenital blind subjects participate in auditory selective attention (Weaver & Stevens, 2007). Several human studies also show that blind subjects are better than sighted subjects at auditory discrimination functions (Kujala et al., 2000).

It was commonly believed that the cross-modal reorganization of brain function in sensory deprivation can only occur in an immature brain (Cohen et al., 1999) but Kujala et al. (1997) suggest that reorganization might occur in across sensory modalities even in adults. Buchel and colleagues suggest that cross-modal plasticity is depends on onset of blindness (Burton, 2003b). So, the aim of our study was to compare amplitude and latency of cognitive potential P3 with high and low frequency stimulus in early and late onset blind subjects to study the neural plasticity based on onset of blindness.

2. Method

15 early (< 6 years) and 15 late (> 12years) onset blind subjects participated in our studies. Early blind subjects were 8 male and 7 female with mean age of 25.13 ± 4.27 years; all but 2 were right handed and late onset blind subjects were 6 male and 9 female with mean age of 27.20 ± 3.21 years; all but 4 were right handed. All blind subjects tested in electrophysiologic clinic in Tehran University of Medical Sciences. In all cases, blindness was due to peripheral deficits. 7 early onset blind subjects had a retinopathy of prematurity, 4 had deficit of the nervous opticus and 3 suffered from a bilateral glaucoma and 1 suffered from an accident. Late blindness originated either from retinophatia pigmentosa, a deficit of the nervous opticus a stargardt or the loss of both eyes due to accident. All blind subjects had a high school degree and normal hearing. The P3 was recorded with oddball paradigm which 2 different stimuli (standard stimulus (80%) and rare stimulus (20%)) were presented in an unpredictable sequence and in which the subject had to press the button when he/she heard a rare stimulus defined as target. The stimuli presented at 70 dBnHl in both groups high frequency (1000/2000 Hz) and low frequency (250/500 Hz).

The P3 was recorded with alternative polarity with Ag/Agcl electrodes from F_Z and C_Z leads with earlobes shunted by a 5 KΩ, the resistor as reference that are connected together with jumper. Fp_Z served as ground electrode. Band pass filter was set 0.1 – 30 Hz. Stimulus locked and random eye movements (blinks) in the blind subjects were suppressed by covering the eye with bandage.

Independent *t*-test, paired *t*-test and Mann-Whitney U test were used for statistical evaluation of the results.

Because of the P3 response has not significant difference between male and female we do not study gender. We omitted effect of handedness with using from C_Z electrode.

3. Results

The sample of P3 responses with high and low frequency in early and late onset blind subjects is shown in fig 1 and fig 2, respectively.

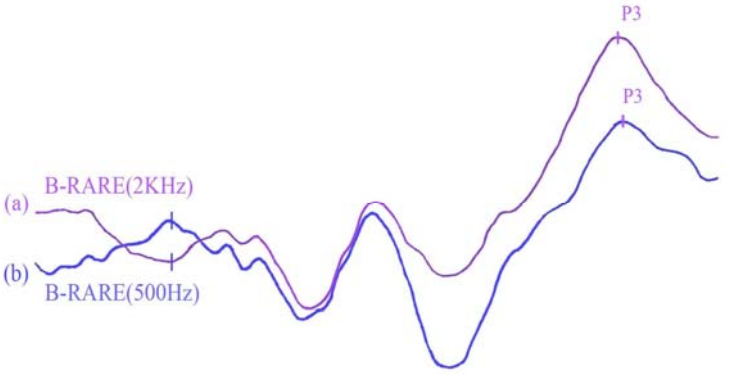

Fig 1. The sample of P3 responses of early onset blind subjects with high (a) and low (b) frequency stimuli

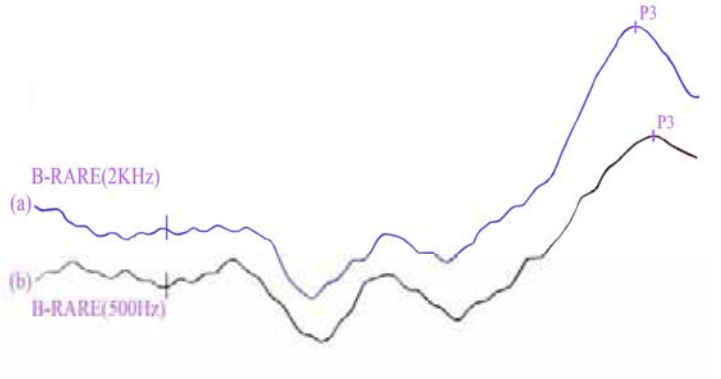

Fig 2. The sample of P3 responses of late onset blind subjects with high (a) and low (b) frequency stimuli

With high frequency stimulus mean amplitude was 14.13 ± 5.53 μV in early and was 11.58 ± 6.61 μV in late onset blind subjects (table 1).

Table1. P3 response domain comparison of amplitude in early and late blinds with high frequency

Group	Average	Standard Deviation	Confidence Interval (90%)		*p*-value
			Lower limit	Upper limit	
Early onset Blind	14.13	5.53	-2.00	7.11	.261
Late onset Blind	11.58	6.61			

Mean latency of P3 obtained 295.60 ± 31.33 ms and 308.38 ± 20.84 ms in early and late blind, respectively. With low frequency stimuli, mean amplitude obtained 17.59 ± 8.17 μV in early onset blind subjects and was 10.50 ± 4.34 μv in late onset (table 2). Mean latency of P3 was 317.38 ± 21.71 ms and 327.42 ± 27.31 ms in early and late onset blind subjects, respectively.

Table 2. P3 response domain comparison of amplitude in early and late blinds with low frequency

Group	Average	Standard Deviation	Confidence Interval (90%)		*p*-value
			Lower limit	Upper limit	
Early onset Blind	17.59	8.17	2.16	12.02	.007
Late onset Blind	10.50	4.34			

Comparison of results between two groups showed that there were no statistically significant differences between amplitudes ($p = 0.26$) and latencies ($p = 0.19$) of P3 with high frequency stimuli, whereas, in low frequency the difference between amplitudes was significant ($p = 0.007$), although there was no significant difference between latencies ($p = 0.21$).

4. Discussion

P3 amplitude in early blind is larger than that in late blind subjects with high frequency stimuli, but this difference is not statistically significant. Kujala et al. (1997) suggest that there is no significant difference between the two groups. Kujala et al. (1997) studied cross-modal plasticity in early and late onset blind subjects and sighted subjects using auditory evoked potential with high-frequency stimulus. When the participant did not attend to auditory stimulus, MMN recorded in each of three groups, but no significant difference was found between the early and late in the anterior-posterior scalp distribution of brain activity. However, when rare stimulus had to be detected, N2 and P3 components were elicited, where scalp distribution in the blind was more posterior than to that in the sighted. This result suggests that cross-modal reorganization also may occur in adults (Kujal et al., 1997). Alho and colleagues have reported that the posterior brain areas of the blind might be activated by auditory stimulus selection when the subject attends selectively to the target auditory stimulus (Alho et al., 1993). Recent studies show that plasticity changes occur in sensory modalities after childhood. When two groups of blind subjects (early and late) have compared with together, maximum electrical activation in response to higher frequency stimulus (target tone) in the blind to that in the sighted that might show reorganization of the brain in adults (Kujala et al., 2000).

Amplitude of P3 response in early onset blind subjects obtained 17.59 μV and obtained 10.50 μV in late onset blind subjects with low frequency is shown that amplitude of P3 is larger than in the early onset blind subjects to that in the late onset blind subjects and this difference is statistically significant ($p = 0.007$).

Elbert et al. (2002) explained hypothesis about that how tonotopic map of auditory cortex has enlarged in blind subjects. He reported that enlargement of auditory cortex in blind subjects is due to reorganization and depends to activity in cortex that occurs either to activate previous silent connections or to establish new neural tracts. It seems that development of auditory cortical area includes increase participating neurons and also, increase bipolar times and there is probably tuning frequency neurons has been more specific. So, a tone with one specific frequency can activate small complex of neurons. However, this mechanism may be separately or overlapped together. So, increase participating neurons have lead to amplitude increase (Elbert et al., 2002). In other studies that have done by other researchers to study P3 in blind subjects have used high frequency stimuli but in our study, in addition to high frequency stimulus, we used low frequency stimulus for recording of P3 in both groups. The significant difference of the this comparison suggest that maybe reorganization depends on onset of blindness (Buchel, Price, Frackowiak, & Friston, 1998). So, visual experience is significant for plasticity of the visual cortex (Price, Ferrer, Blakemore, & Kato, 1994; Blakemore, 1991). In spite of studies that show effect of visual experience in visual cortex functional development, others obtained with high frequency stimuli (shown in this study) have indicated that there is no significant between activation patterns and onset of blindness in early and late onset blind subjects (Buchel et al.,

1998). Whereas, Kujala et al. (2000) reported that in spite of occurrence of plastic changes after puberty but activation pattern between two groups is different. Kujala demonstrated that in early-blind, only the extra-striate cortex was activated, but in the late onset blind, both the striate- and extra-striate cortices were activated (Kujala et al., 2000). So, it seems, due to difference activation a pattern in visual cortex, this difference was shown better by low frequency stimulus.

In our study, P3 latency obtained 295.60ms in early blind and 303.38ms in late-blind with high frequency stimulus and obtained 317.38 ms in early and 327.42 ms in late blind with low frequency that was statistically no significant ($p = 0.19$ and $p = 0.21$), respectively that agree to the results cited above. In the other hands, between P3 latency in early and late onset blind subjects was no significant difference with low frequency stimulus in the same condition. In our study, age of onset of blindness was not lead to decrease latency of cognitive potential in late onset blind subjects. Some studies indicate V1 activation in early onset blind subjects (Sadato et al., 1998, 1996) and others most show V1 activation in late blind (Buchel et al., 1998). Burton et al. (2003a) reported this activation in all of section of visual cortex in all of blind individuals while Braille reading without attention to age of onset of blindness (Burton, 2003b). Cortical imaging suggests that multi-sensory changes occur in all of life, because early and late blind subjects showed comparable changes. However, reorganization in both groups is not similar together. So, it seems, maturation and visual experience can affect (Burton, 2003b).

5. Conclusion

There were no significant differences between amplitude and latency with high frequency stimuli, though there was significant difference between amplitude with low frequency. Neuroplasticity seemed to occur in visual cortex in both groups, which suggested that it might not depend on the onset age of deprivations. It appeared that activation patterns of the occipital cortex differed between these two groups and this could be shown with low frequency stimuli better than with high frequency stimuli.

Acknowledgement

With thanks to Mr. Ma'aroufi, president of "Asaye Sefid" Institute, Ms. Asghari, manager of the Iranian Blind Scientific Association, "Farhangsaraye Salmand" and "Farhangsaraye Bahman" for their kind helps in our research and special thanks for Hossein H. Ebrahimi.

References

Alho, K., Kujala, T., Paavilainen, P., Summala, H., & Naatanen, R. (1993). Auditory processing in visual brain areas of the early blind: Evidence from event-related potentials. *Electroencephalography and Clinical Neurophysiology, 86,* 418-427.

Blakemore, C. (1991). Sensitive and vulnerable periods in the development of the visual system. *Ciba Foundation Symposium, 156,* 129-147.

Buchel, C., Price, C., Frackowiak, R. S., & Friston, K. (1998). Different activation patterns in the visual cortex of late and congenitally blind subjects. *Brain, 121,* 409-419.

Burton, H., Diamond, J., & McDermott, K. (2003a). dissociating cortical regions activated by semantic and phonological tasks: A FMRI study in blind and sighted people. *Journal of Neurophysiology, 90*, 1965-1982.

Burton, H. (2003b). Visual cortex activity in early and late blind people. *Journal of Neuroscience, 23*, 4005-4011.

Cohen, L. G., Weeks, R. A., Sadato, N., Celnik, P., Ishii, K., & Hallett, M. (1999). Period of susceptibility for cross-modal plasticity in the blind. *Annals of Neurology, 45*, 451-460.

Elbert, T., Sterr, A., Rockstroh, B., Pantev, C., Muller, M. M., & Taub, E. (2002). Expansion of the tonotopic area in the auditory cortex of the blind. *Journal of Neuroscience, 22*, 9941-9944.

Hall, J. W. (2007). P300 response. In J. W. Hall III (Ed.), New Handbook of Auditory evoked responses (pp. 518-548). Pearson.

Kujala, T., Alho, K., Huotilainen, M., Ilmoniemi, R. J., Lehtokoski, A., Leinonen, A., et al. (1997). Electrophysiological evidence for cross-modal plasticity in humans with early- and late-onset blindness. *Psychophysiology, 34*, 213-216.

Kujala, T., Alho, K., & Naatanen, R. (2000). Cross-modal reorganization of human cortical functions. *Trends in Neurosciences, 23*, 115-120.

Leclerc, C., Saint-Amour, D., Lavoie, M., Lassonde, M., & Lepore, F. (2000). Brain functional reorganization in early blind humans revealed by auditory event-related potentials. *Neuroreport, 11*, 545-550.

Maurer, D., Lewis, T. L., & Mondloch, C. J. (2005). Missing sights: Consequences for visual cognitive development. *Trends in Cognitive Sciences, 9*, 144-151.

Naveen, K. V., Srinivas, R. S., Nirmala, K. S., Nagendra, H. R., & Telles, S. (1997). Middle latency auditory evoked potentials in congenitally blind and normal sighted subjects. *International Journal of Neuroscience, 90*, 105-111.

Niemeyer, W., & Starlinger, I. (1981). Do the blind hear better? Investigations on auditory processing in congenital or early acquired blindness. II. Central functions. *Audiology, 20*, 510-515.

Price, D. J., Ferrer, J. M., Blakemore, C., & Kato, N. (1994). Postnatal development and plasticity of corticocortical projections from area 17 to area 18 in the cat's visual cortex. *Journal of Neuroscience, 14*, 2747-2762.

Roder, B., Rosler, F., Hennighausen, E., & Nacker, F. (1996). Event-related potentials during auditory and somatosensory discrimination in sighted and blind human subjects. *Brain Researc, Cognitive Brain Research, 4*, 77-93.

Roder, B., Rosler, F., & Neville, H. J. (2000). Event-related potentials during auditory language processing in congenitally blind and sighted people. *Neuropsychologia, 38*, 1482-1502.

Sadato, N., Pascual-Leone, A., Grafman, J., Deiber, M. P., Ibanez, V., & Hallett, M. (1998). Neural networks for Braille reading by the blind. *Brain, 121*, 1213-1229.

Sadato, N., Pascual-Leone, A., Grafman, J., Ibanez, V., Deiber, M. P., Dold, G., et al. (1996). Activation of the primary visual cortex by Braille reading in blind subjects. *Nature, 380*, 526-528.

Stevens, A. A. & Weaver, K. E. (2009). Functional characteristics of auditory cortex in the blind. *Behavioral Brain Research, 196*, 134-138.

Theoret, H., Merabet, L., & Pascual-Leone, A. (2004). Behavioral and neuroplastic changes in the blind: Evidence for functionally relevant cross-modal interactions. *Journal of Physiology-Paris, 98*, 221-233.

Weaver, K., & Stevens, A. (2007). Attention and sensory interactions within the occipital cortex in the early blind: An FMRI study. *Journal of Cognitive Neuroscience, 19*, 315-330.

Woods, D. L., Clayworth, C. C., & Bach-Y-Rita, P. (1995). *Early blindness reorganizes auditory processing in humans*. Paper presented at the meeting of the 15th annual meeting, Dallas, TX.